AEPCOS QUARTERLY PUBLICATION LIST
APRIL - JUNE 2015

Highlighted articles

Wang F, Pan J, Liu Y, Meng Q, Lv P, Qu F, Ding GL, Klausen C, Leung PCK, Chan HC, Yao W, Zhou CY, Shi B, Zhang J, Sheng J, Huang H. Alternative splicing of the androgen receptor in polycystic ovary syndrome. PNAS April, 2015;112(15): 4743-4748.

The authors identified two alternative splice variants (ASVs) of the androgen receptor (AR), an insertion into intron 2 (ins isoform) and a deletion in exon 3 (del isoform), in granulosa cells (GCs) of some PCOS patients. They found these ASVs of AR in 62% out of the 68 PCOS patients and not in any of the 120 control women. The ASVs of AR were associated with hyperandrogenism and abnormalities in folliculogenesis. Alternative splicing altered genome-wide AR recruitment and androgen induced expression of genes related to androgen metabolism and folliculogenesis in GCs. The clinical evaluation revealed that both ins and del AR were associated with higher androgen levels in serum and follicles. Also, ins was linked to longer cycles and a greater number of antral follicles in PCOS patients. Consistently, ins was also associated with higher follicular fluid levels of anti- Müllerian hormone. These findings establish alternative splicing of AR in GCs as a major pathogenic mechanism for hyperandrogenism and abnormal folliculogenesis in PCOS. However, these findings have to be reproduced and confirmed in other studies and other populations. (CM)

Greenwood EA, Pasch LA, Shinkai K, Cedars MI, Huddleston HG. Putative role for insulin resistance in depression risk in polycystic ovary syndrome. FertilSteril. 2015 Sep;104(3):707-14.e1. doi: 10.1016/j.fertnstert.2015.05.019. Epub 2015 Jun 19. PubMed PMID: 26054555.

There is increased recognition that PCOS may be associated with mood disorders with increased prevalence of depression and anxiety seen. It is not clear however what the mechanisms may be for this and if they are related to metabolic abnormalities. Obesity is also known to play a role in depression and the high prevalence of obesity in PCOS makes the analysis of independent factors more complex. In this cross-sectional study of 301 women of reproductive age with PCOS, the authors evaluated the Beck Depression Inventory Fast Screen (BDI-FS) with physical and metabolic findings in PCOS. Of the 301 women with PCOS, 131 were found to be at risk for depression as determined by a positive BDI-FS screen (score >4). This association was influenced by BMI with 34% of lean, 43% of overweight and 50% of obese women demonstrating positive screen for depression, with an OR of 3.0 (1.74-5.01) for depression in obese compared to lean women. When the authors stratified the analysis by BMI, obese women with higher HOMA-IR had significantly higher risk of depression that similarly matched obese women with lower HOMA-IR. In a multivariate logistic regression, HOMA-IR was independently, although modestly) related to the risk of depression after controlling for BMI, age, ethnicity and exercise. This relationship however was not

seen in the lean women with PCOS suggesting that it is the interplay with obesity that may be important. Androgen excess was not associated with different BDI-FS score. Additionally this relationship between HOMA-IR and BDI-FS was seen in mild and moderate depression scores but not severe depression, suggesting other mechanisms in women with severe depression. This study is limited by only the screen for depression, not a follow up interview and the use of HOMA-IR as a measure of insulin resistance but these tools are available easily for large scale screening of populations. Overall this study serves to again emphasize that mental health screening should be a routine assessment in the care of women with PCOS given the high prevalence of abnormal screening. It also implicates the association with abnormal metabolic parameters, specifically the measure of insulin resistance using the homeostasis model of assessment of insulin resistance as a potential contributor to mild and moderate depression risk in PCOS. Further investigations, with larger cohorts, are needed to clarify this association. (KMH)

Caldwell AS, Eid S, Kay CR, Jimenez M, McMahon AC, Desai R, Allan CM, Smith JT, Handelsman DJ, Walters KA. Haplosufficient genomic androgen receptor signaling is adequate to protect female mice from induction of polycystic ovary syndrome features by prenatal hyperandrogenization. Endocrinology. 2015 Apr;156(4):1441-52. doi: 10.1210/en.2014-1887. Epub 2015 Feb 2. PubMed PMID: 25643156.

This pathogenic mechanism study used wild-type (WT) and androgen receptor (AR) knockout (ARKO) mice, together with a mouse model of PCOS, to investigate the contribution of genomic AR-mediated actions in the development of PCOS-like traits in these female rodents. PCOS-like features were induced by prenatal exposure to dihydrotestosterone on days 16-18 of gestation in WT, heterozygote, and homozygote ARKO mice. DHT treatment of WT mice induced ovarian cysts, disrupted estrous cycles (42% vs 100% cycling), and led to ~50% fewer corpora lutea. Diestrus serum LH and FSH, and estradiol-induced-negative feedback as well as hypothalamic expression of kisspeptin, neurokinin B, and dynorphin, however, were not rendered PCOS-like by DHT treatment in WT mice. DHT-treated WT mice exhibited a more than 48% increase in adipocyte area, but without changes in body fat. In contrast, heterozygous and homozygous ARKO mice exposed to DHT maintained comparable and normal ovarian morphology, estrous cycling, and corpora lutea numbers, without any increase in adipocyte size. These findings provide compelling evidence that genomic AR signaling is an important mediator in the development of prenatal androgen mediated PCOS-like traits with a dose dependency that allows even AR haplosufficiency to prevent induction by prenatal androgenization of PCOS features in adult life. Thus a full complement of functional AR are required for prenatal androgen pathogenic origins of PCOS-like traits, and estrogen receptors play secondary roles in this pathogenic mechanism.(DA)

Comim FV, Hardy K, Robinson J, Franks S. Disorders of follicle development and steroidogenesis in ovaries of androgenised foetal sheep. J Endocrinol. 2015 Apr;225(1):39-46. doi: 10.1530/JOE-14-0150. PubMed PMID: 25792297.

This study used a PCOS sheep model to examine evidence for precocious primordial follicle formation at day 90 of gestation in ovaries of fetal Poll Dorset sheep exposed to testosterone (T) excess during early-to-mid gestation. Using a specific marker of germ cells (VASA homologue protein) in ovarian sections, the authors found that T-exposed sheep had nearly double the proportion of germ cells enclosed in follicles compared with controls. When analyzed by follicle stage, there was no significant difference between groups in the proportion of primordial follicles and growing (transitional and primary) follicles. Differences between T-exposed and control fetal sheep were found in both mRNA and in protein expression of steroidogenic enzymes and androgen receptor. These results in Dorset ewes were not only complementary to previous reports, but also suggested that the timing of follicle formation and steroidogenic activity may vary between different breeds as well as in response to fetal T exposure. These data show that granulosa cells constitute a specific target for programming by T in utero. Given that there is an increase in the density of preantral follicles in women with polycystic ovaries, these sheep findings suggest that PCOS-like aberrant follicle development is programmed by exposure of the ovary to excess T during fetal life.(DA)

List of Publications

*Publications were searched in pubmed with primary search criteria congenital adrenal hyperplasia, premature adrenarche or PCOS with secondary subcategory, inclusive of the quarter dates. Every attempt was made to include all papers in English in these categories but may not be an exhaustive list. If a related paper was published in this quarter and was inadvertently not included, please notify the publications committee so that we may include in the following quarterly review

Congenital Adrenal Hyperplasia and Disorders of Steroidogenesis

Ambroziak U, Kępczyńska-Nyk A, Kuryłowicz A, Wysłouch-Cieszyńska A, Małunowicz EM, Bartoszewicz Z, Kondracka A, Jaźwiec R, Pawłowska E, Szcześniak M, Dadlez M, Bednarczuk T. LC-MS/MS improves screening towards 21-hydroxylase deficiency. Gynecol Endocrinol. 2015 Apr;31(4):296-300. doi: 10.3109/09513590.2014.994599. Epub 2014 Dec 24. PubMed PMID: 25539143.

Auchus RJ. Management considerations for the adult with congenital adrenal hyperplasia. Mol Cell Endocrinol. 2015 Jun 15;408:190-7. doi: 10.1016/j.mce.2015.01.039. Epub 2015 Jan 30. Review. PubMed PMID: 25643980.

Auchus RJ. The classic and nonclassic concenital adrenal hyperplasias. Endocr Pract. 2015 Apr;21(4):383-9. doi: 10.4158/EP14474.RA. Epub 2014 Dec 22. PubMed PMID: 25536973.

Bloem LM, Storbeck KH, Swart P, du Toit T, Schloms L, Swart AC. Advances in the analytical methodologies: Profiling steroids in familiar pathways-challenging

dogmas. J Steroid Biochem Mol Biol. 2015 Sep;153:80-92. doi: 10.1016/j.jsbmb.2015.04.009. Epub 2015 Apr 11. Review. PubMed PMID: 25869556.

Bonfig W, Roehl FW, Riedl S, Dörr HG, Bettendorf M, Brämswig J, Schönau E, Riepe F, Hauffa B, Holl RW, Mohnike K; AQUAPE CAH Study Group. Blood Pressure in a Large Cohort of Children and Adolescents With Classic Adrenal Hyperplasia (CAH) Due to 21-Hydroxylase Deficiency. Am J Hypertens. 2015 Jun 11. pii: hpv087. [Epub ahead of print] PubMed PMID: 26071487.

Brett EM, Auchus RJ. Genetic forms of adrenal insufficiency. Endocr Pract. 2015 Apr;21(4):395-9. doi: 10.4158/EP14503.RA. Epub 2015 Feb 9. PubMed PMID: 25667374.

Delle Piane L, Rinaudo PF, Miller WL. 150 years of congenital adrenal hyperplasia: translation and commentary of De Crecchio's classic paper from 1865. Endocrinology. 2015 Apr;156(4):1210-7. doi: 10.1210/en.2014-1879. Epub 2015 Jan 30. PubMed PMID: 25635623.

Delvecchio M, Soldano L, Lonero A, Ventura A, Giordano P, Cavallo L, Grano M, Brunetti G, Faienza MF. Evaluation of impact of steroid replacement treatment on bone health in children with 21-hydroxylase deficiency. Endocrine. 2015 Apr;48(3):995-1000. doi: 10.1007/s12020-014-0332-9. Epub 2014 Jul 1. PubMed PMID: 24981037.

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Hampson E, Rovet JF. Spatial function in adolescents and young adults with congenital adrenal hyperplasia: clinical phenotype and implications for the androgen hypothesis. Psychoneuroendocrinology. 2015 Apr;54:60-70. doi: 10.1016/j.psyneuen.2015.01.022. Epub 2015 Feb 2. PubMed PMID: 25686803.

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Hindmarsh PC, Charmandari E. Variation in absorption and half-life of hydrocortisone influence plasma cortisol concentrations. Clin Endocrinol (Oxf). 2015 Apr;82(4):557-61. doi: 10.1111/cen.12653. Epub 2014 Dec 19. PubMed PMID: 25369980.

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Magiakou AM, Kanaka-Gantenbein C, Chrousos GP, Dacou-Voutetakis C. The spectrum of clinical, hormonal and molecular findings in 280 individuals with nonclassical congenital adrenal hyperplasia caused by mutations of the CYP21A2 gene. Clin Endocrinol (Oxf). 2015 Apr;82(4):543-9. doi: 10.1111/cen.12543. Epub 2014 Aug 3. PubMed PMID: 25041270.

Miller WL. Fetal endocrine therapy for congenital adrenal hyperplasia should not be done. Best Pract Res Clin Endocrinol Metab. 2015 Jun;29(3):469-83. doi: 10.1016/j.beem.2015.01.005. Epub 2015 Jan 24. Review. PubMed PMID: 26051303.

Mooij CF, Parajes S, Pijnenburg-Kleizen KJ, Arlt W, Krone N, Claahsen-van der Grinten HL. Influence of 17-Hydroxyprogesterone, Progesterone and Sex Steroids on Mineralocorticoid Receptor Transactivation in Congenital Adrenal Hyperplasia. Horm Res Paediatr. 2015 Apr 15. [Epub ahead of print] PubMed PMID: 25896481.

Muñoz LN, Ochetti M, Perez G, Sobrero GM, Silvano LK, Martin SE, Testa GM, Miras MB. Measurement of Serum 17α-Hydroxyprogesterone in Infants by Radioimmunoassay. Pediatr Endocrinol Rev. 2015 Jun;12(4):366-72. PubMed PMID: 26182481.

Pallan PS, Wang C, Lei L, Yoshimoto FK, Auchus RJ, Waterman MR, Guengerich FP, Egli M. Human Cytochrome P450 21A2, the Major Steroid 21-Hydroxylase: STRUCTURE OF THE ENZYME·PROGESTERONE SUBSTRATE COMPLEX AND RATE-LIMITING C-H BOND CLEAVAGE. J Biol Chem. 2015 May 22;290(21):13128-43. doi: 10.1074/jbc.M115.646307. Epub 2015 Apr 8. PubMed PMID: 25855791; PubMed Central PMCID: PMC4505568.

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Pina C, Khattab A, Katzman P, Bruckner L, Andolina J, New M, Yau M. Ovarian carcinoma in a 14-year-old with classical salt-wasting congenital adrenal hyperplasia and bilateral adrenalectomy. J Pediatr Endocrinol Metab. 2015 May;28(5-6):663-7. doi: 10.1515/jpem-2014-0299. PubMed PMID: 25427061.

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PMC4506691.

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Webb EA, Krone N. Current and novel approaches to children and young people with congenital adrenal hyperplasia and adrenal insufficiency. Best Pract Res Clin Endocrinol Metab. 2015 Jun;29(3):449-68. doi: 10.1016/j.beem.2015.04.002. Epub 2015 Apr 22. Review. PubMed PMID: 26051302.

Premature adrenarche

de Melo AS, Dias SV, Cavalli Rde C, Cardoso VC, Bettiol H, Barbieri MA, Ferriani RA, Vieira CS. Pathogenesis of polycystic ovary syndrome: multifactorial assessment from the foetal stage to menopause. Reproduction. 2015 Jul;150(1):R11-24. doi: 10.1530/REP-14-0499. Epub 2015 Apr 2. Review. PubMed PMID: 25835506.

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PCOS-Adolescence

Akın L, Kendirci M, Narin F, Kurtoglu S, Saraymen R, Kondolot M, Koçak S, Elmali F. The endocrine disruptor bisphenol A may play a role in the aetiopathogenesis of polycystic ovary syndrome in adolescent girls. Acta Paediatr. 2015 Apr;104(4):e171-7. doi: 10.1111/apa.12885. Epub 2015 Feb 3. PubMed PMID: 25469562.

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PubMed PMID: 25430596.

Fruzzetti F, Campagna AM, Perini D, Carmina E. Ovarian volume in normal and hyperandrogenic adolescent women. Fertil Steril. 2015 Jul;104(1):196-9. doi: 10.1016/j.fertnstert.2015.03.026. Epub 2015 Apr 29. PubMed PMID: 25934594.

Glintborg D, Hass Rubin K, Nybo M, Abrahamsen B, Andersen M. Morbidity and medicine prescriptions in a nationwide Danish population of patients diagnosed with polycystic ovary syndrome. Eur J Endocrinol. 2015 May;172(5):627-38. doi: 10.1530/EJE-14-1108. Epub 2015 Feb 5. PubMed PMID: 25656495.

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PCOS-Dermatology

Moro F, Tropea A, Scarinci E, Federico A, De Simone C, Caldarola G, Leoncini E, Boccia S, Lanzone A, Apa R. Psoriasis and polycystic ovary syndrome: a new link in different phenotypes. Eur J Obstet Gynecol Reprod Biol. 2015 Aug;191:101-5. doi: 10.1016/j.ejogrb.2015.06.002. Epub 2015 Jun 16. PubMed PMID: 26115053.

PCOS-Endocrine Disruptors

Akın L, Kendirci M, Narin F, Kurtoglu S, Saraymen R, Kondolot M, Koçak S, Elmali F. The endocrine disruptor bisphenol A may play a role in the aetiopathogenesis of polycystic ovary syndrome in adolescent girls. Acta Paediatr. 2015 Apr;104(4):e171-7. doi: 10.1111/apa.12885. Epub 2015 Feb 3. PubMed PMID: 25469562.

PCOS-Animal models

Caldwell AS, Eid S, Kay CR, Jimenez M, McMahon AC, Desai R, Allan CM, Smith JT, Handelsman DJ, Walters KA. Haplosufficient genomic androgen receptor signaling is adequate to protect female mice from induction of polycystic ovary syndrome features by prenatal hyperandrogenization. Endocrinology. 2015 Apr;156(4):1441-52. doi: 10.1210/en.2014-1887. Epub 2015 Feb 2. PubMed PMID: 25643156.

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Programming of Reproductive Neuroendocrine, Ovarian and Metabolic Dysfunctions. Neuroendocrinology. 2015;102(3):226-37. doi: 10.1159/000381830. Epub 2015 Apr 1. PubMed PMID: 25832114; PubMed Central PMCID: PMC4591099.

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Ongaro L, Salvetti NR, Giovambattista A, Spinedi E, Ortega HH. Neonatal androgenization-induced early endocrine-metabolic and ovary misprogramming in the female rat. Life Sci. 2015 Jun 1;130:66-72. doi: 10.1016/j.lfs.2015.03.008. Epub 2015 Mar 26. PubMed PMID: 25818182.

Ressler IB, Grayson BE, Ulrich-Lai YM, Seeley RJ. Diet-induced obesity exacerbates metabolic and behavioral effects of polycystic ovary syndrome in a rodent model. Am J Physiol Endocrinol Metab. 2015 Jun 15;308(12):E1076-84. doi: 10.1152/ajpendo.00182.2014. Epub 2015 Apr 14. PubMed PMID: 26078189; PubMed Central PMCID: PMC4469809.

Salilew-Wondim D, Wang Q, Tesfaye D, Schellander K, Hoelker M, Hossain MM, Tsang BK. Polycystic ovarian syndrome is accompanied by repression of gene signatures associated with biosynthesis and metabolism of steroids, cholesterol and lipids. J Ovarian Res. 2015 Apr 13;8(1):24. doi: 10.1186/s13048-015-0151-5. PubMed PMID: 25887459; PubMed Central PMCID: PMC4414284.

PCOS-General Health

Doherty DA, Newnham JP, Bower C, Hart R. Implications of polycystic ovary syndrome for pregnancy and for the health of offspring. Obstet Gynecol. 2015 Jun;125(6):1397-406. doi: 10.1097/AOG.0000000000000852. PubMed PMID: 26000511.

Joham AE, Teede HJ, Ranasinha S, Zoungas S, Boyle J. Prevalence of infertility

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PCOS - Genetics

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